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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/931,375	08/17/2001	Matthew L. Warman	38464-0004	1602
24024	7590	10/26/2005	EXAMINER	
CALFEE HALTER & GRISWOLD, LLP 800 SUPERIOR AVENUE SUITE 1400 CLEVELAND, OH 44114			SEHARASEYON, JEGATHEESAN	
			ART UNIT	PAPER NUMBER
			1647	

DATE MAILED: 10/26/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/931,375	WARMAN ET AL.	
	Examiner	Art Unit	
	Jegatheesan Seharaseyon, Ph.D	1647	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
 - If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
 - Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) Responsive to communication(s) filed on 08 August 2005.
- 2a) This action is FINAL. 2b) This action is non-final.
- 3) Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) Claim(s) 8,9,30 and 32-38 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) Claim(s) _____ is/are allowed.
- 6) Claim(s) 8,9,30 and 32-38 is/are rejected.
- 7) Claim(s) _____ is/are objected to.
- 8) Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) The specification is objected to by the Examiner.
- 10) The drawing(s) filed on _____ is/are: a) accepted or b) objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) All b) Some * c) None of:
 1. Certified copies of the priority documents have been received.
 2. Certified copies of the priority documents have been received in Application No. _____.
 3. Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) Notice of References Cited (PTO-892)
- 2) Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)
Paper No(s)/Mail Date _____
- 4) Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____
- 5) Notice of Informal Patent Application (PTO-152)
- 6) Other: _____

DETAILED ACTION

1. This office action is in response to the amendment and remarks filed on 8/8/04. Claim 31 has been cancelled. Claims 8, 9 and 30 are amended. Claims 37 and 38 have been newly added. Thus, claims 8, 9, 30 and 32-38 are pending.
2. The text of those sections of Title 35, U. S. Code not included in this action can be found in a prior Office action.
3. The Office also acknowledges the change in the specification.
4. The Office also acknowledges the changes to the Figures.

Claim Objections

5. Applicants amendments have necessitated the withdrawal of the objection to claim 8.
6. Claims 32 and 38 are objected to because it is dependent on cancelled claim. The Office is assuming that claim 32 is dependent on claim 30 and examined further. Appropriate correction is required.

Claim Rejections - 35 USC § 112

7. The rejection of claims 8,9 and 30 under 35 U.S.C 112, second paragraph, as being indefinite for failing to point out distinctly the subject matter is withdrawn because of Applicants amendments.
8. The rejection of claim 35 for lacking antecedent basis is maintained for reason stated in the Office Action dated 3/18/2005 (see page 5).

Claim Rejections - 35 USC § 102

9. Applicants amendments necessitated the withdrawal of the rejection of claims 8, 30 and 31 under 35 U.S.C 102(e) as being anticipated by Carulli et al.

Claim Rejections - 35 USC § 103

10. The pending rejections of claims 30-36 under 35 U.S.C 103(a) are withdrawn because of Applicants amendments. Applicants' arguments are moot because Applicants have modified the claims. Office notes that the receptor binding of various molecules is an inherent function of any receptor including the instant receptor (BSMR).

11. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action.

12. Claims 8, 9, 30, 32, 37 and 38 are rejected under 35 U.S.C. 103(a) as being unpatentable over Carulli et al. (U. S. Patent NO. 6, 780, 609) and Dong et al. (1998, Ref B06 in PTO1449 of 10/15/02) in view of Tamai et al. (2000, Ref A24 in PTO1449 of 5/1/02).

The teachings of Carulli et al. have been discussed in the Office Action dated 3/18/2005 paragraph 9a and above in paragraph 10. The Zmax1 or HBM protein involved in the regulation of bone strength and mineralization disclosed by Carulli et al. (SEQ ID NO: 3) is different from SEQ ID NO: 2 of the instant invention at 3 positions possibly due to sequencing errors. Thus, the Office provides Dong et al., which is identical to SEQ ID NO: 2 (see Appendix A). This reference further teaches the mitogenic activity of this protein in osteoblastic cell line TE85 (see page 788). It is further suggested that this gene expression is regulated during osteoblast differentiation (see page 789). However, these references do no teach the modulation of this gene by WNT signaling. Tamai et al. (2000) describe that human LRP5 Human LRP5 and LRP6 share 71% amino-acid identity and together with Arrow, form a distinct subgroup of the LRP

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family. Arrow, LRP5 and LRP6 each contain an extracellular domain with a EGF (epidermal growth factor) repeats and LDLR repeats, followed by a transmembrane region and a cytoplasmic domain lacking recognizable catalytic motifs. Tamai et al. study the LRP5/LRP6 involvement in Wnt signaling by examining their function in Wnt-induced axis and neural crest formation in *Xenopus* embryos. It also teaches that although LRP5 alone did not induce axes, co-injecting LRP5 and Wnt-5a did induce axes (see page 531). Tamai et al. reference also discloses the induction LRP6 by Wnt-1 and Wnt-3a (see page 532).

It would have been obvious to one of ordinary skill in the art at the time the invention was made to use BSMR protein to modulate bone strength and mineralization as described by Carulli et al and Dong et al. using WNT proteins because Tamai et al. disclose that LRP5 is induced by WNT signaling. One of ordinary skill in the art would have been motivated to modulate BSMR (LRP5 or Zmax1 or LR3) using WNT signaling in order to regulate bone strength and mineralization. In addition, one of ordinary skill in the art would have been also been motivated because Tamai et al. describe the signal transduction of LRP5/LRP6 by Wnt (page 531). Therefore, the instant invention is *prima facie* obvious over Carulli et al. (U. S. Patent NO. 6, 780, 609) and Dong et al. (1998, Ref B06 in PTO1449 of 10/15/02) in view of Tamai et al. (2000, Ref A24 in PTO1449 of 5/1/02).

13. Claims 33 and 34 are rejected under 35 U.S.C. 103(a) as being unpatentable over Carulli et al. (U. S. Patent NO. 6, 780, 609) and Dong et al. (1998, Ref B06 in PTO1449

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of 10/15/02) in view of Tamai et al. (2000, Ref A24 in PTO1449 of 5/1/02) and Oppermann et al. (U. S. Patent NO. 5, 652, 337).

The teachings of Carulli et al., Dong et al., Tamai et al. and Oppermann et al. have been described above and in the Office Action dated 3/18/2005, paragraph 10b (see page 9).

Specifically, Oppermann et al. disclose compounds that are capable of targeting BSMR effector to the region of bone remodeling (column 15, lines 38-42). For example, tetracycline and diphosphonates (bisphosphonates) are known to bind to bone mineral, particularly at zones of bone remodeling, when they are provided systemically in a mammal. Accordingly, these molecules may be included as useful agents for targeting OP-3 (a morphogen) to bone tissue. Alternatively, an antibody or other binding protein that interacts specifically with a surface molecule on the desired target tissue cells also may be used (column 15, lines 38-47).

It would have been obvious to one of ordinary skill in the art at the time the invention was made to target BSMR effector molecules to regions of bone regeneration or remodeling to modulate bone strength and mineralization as described by Carulli et al., Dong et al. and Tamai et al. collectively using tetracycline and diphosphonates (bisphosphonates) that are known to bind to bone mineral because Oppermann et al. disclose that tetracycline and diphosphonates (bisphosphonates) are known to bind to bone mineral, particularly at zones of bone remodeling, when they are provided systemically in a mammal. One of ordinary skill in the art would have been motivated to modulate BSMR (LRP5 or Zmax1 or LR3) using a BSMR effector such as Wnt that is

targeted to bone producing or remodeling region by compounds such as tetracycline and diphosphonates (bisphosphonates) in order to regulate bone strength and mineralization to treat osteoporosis. Therefore, the instant invention is *prima facie* obvious over Carulli et al. (U. S. Patent NO. 6, 780, 609) and Dong et al. (1998, Ref B06 in PTO1449 of 10/15/02) in view of Tamai et al. (2000, Ref A24 in PTO1449 of 5/1/02) and Oppermann et al. (U. S. Patent NO. 5, 652, 337).

14. Claims 35 and 36 are rejected under 35 U.S.C. 103(a) as being unpatentable over Carulli et al. (U. S. Patent NO. 6, 780, 609) and Dong et al. (1998, Ref B06 in PTO1449 of 10/15/02) in view of Tamai et al. (2000, Ref A24 in PTO1449 of 5/1/02) further in view of Wang et al. (U. S. Patent NO. 6, 245, 889) and Hughes et al (1995).

The teachings of Carulli et al., Dong et al., Tamai et al., Wang et al. and Hughes et al. have been described above and in the Office Action dated 3/18/2005, paragraph 10c (see page 10).

Wang et al. disclose the use of BMP-2 and BMP-4 protein may be combined with other agents beneficial to the treatment of the bone and/or cartilage defect, wound, or tissue in question (column 6, lines 65 to column 7, lines 42). These agents include various growth factors such as epidermal growth factor (EGF), platelet derived growth factor (PDGF), transforming growth factors (TGF- α and TGF- β), and insulin-like growth factor (IGF) (column 7, lines 2-5). In addition, these agents may provide an environment to attract bone-forming cells, stimulate growth of bone-forming cells or induce differentiation of progenitors of bone forming cells (column 6, lines 20-23). Wang et al. also teach that for bone and/or cartilage formation, the composition would include a

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matrix capable of delivering BMP-2, BMP-4 or other BMP proteins to the site of bone and/or cartilage damage (column 7, lines 35-38). It also teaches that BMP-2 may be used individually in a pharmaceutical composition or in combination with BMP-4 and/or one or more of the other BMP proteins (column 7, lines 14-18). Hughes et al. (1995) disclose that the effect of BMPs on nodule formation was seen after only 24 hours of exposure to BMPs. It also teaches that continuous or 24-h exposure to BMP-2 or BMP-4 increased the number of postmitotic ALP-positive cells in log phase culture (abstract). Thus, it would have been obvious to one of ordinary skill in the art at the time the invention was made to administer another bone morphogenic protein (BMP) target to regions of bone regeneration or remodeling to modulate bone strength and mineralization as described by Wang et al. using a bone morphogenetic protein such as BMP-2 administering BMP-2 at least 24 hrs prior administering the BSMR effector as taught by Hughes et al to increase bone formation because Carulli et al., Dong et al. and Tamai et al. collectively disclose that by manipulating the levels of functional Zmax1 or LRP5 or LR3 protein, it is possible to affect bone development and to increase or decrease levels of bone mineralization, particularly at zones of bone remodeling, when they are provided systemically in a mammal. One of ordinary skill in the art would have been motivated to treat osteoporosis by administering BMP-2 protein 24 hrs prior to providing BSMR effector that is targeted to bone producing or remodeling region in order to regulate bone strength and mineralization to treat osteoporosis. Further, Hughes teaches that BMP-2 or BMP-4 increased the number of postmitotic ALP-positive cells. Therefore, the instant invention is *prima facie* obvious over Carulli et al.

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(U. S. Patent NO. 6, 780, 609) and Dong et al. (1998, Ref B06 in PTO1449 of 10/15/02) in view of Tamai et al. (2000, Ref A24 in PTO1449 of 5/1/02) and Oppermann et al. (U. S. Patent NO. 5, 652, 337).

15. No Claims are allowable.

Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Contact Information

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Jegatheesan Seharaseyon, Ph.D whose telephone number is 571-272-0892. The examiner can normally be reached on M-F: 8:30-5:00.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Brenda Brumback can be reached on 571-272-0961. The fax phone number for the organization where this application or proceeding is assigned is 571-

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273-8300. Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

JSS 10/05



JANET L. ANDRES
SUPERVISORY PATENT EXAMINER

Applicants may

Appendix A

Tue Feb 22 09:16:46 2005

us-09-931-375a-2.rup

Db	121 KLYWTDSERTNIEVANLNGTSRKVLFWQDIDQPRALDPAKSMYTDWGTPRIERAG	Qy	1261 PDPACATGSDICPGAWRGDFRCPDOSDECPVCVAQPCARGCYDLRLRGDE
Qy	181 MDCSRTKLVIVDIDTYWENGLTIDLBOKLMDADAKLSPHTHARLDSFRQKVEGLTHP	Db	1261 PDPACATGSDICPGAWRGDFRCPDOSDECPVCVAQPCARGCYDLRLRGDE
Db	181 MDCSRTKLVIVDIDTYWENGLTIDLBOKLMDADAKLSPHTHARLDSFRQKVEGLTHP	Qy	1241 FALTSGDTWTDWOTRSIMACNRGTGKRRKTSALSYSPDIOVSQSERPFTRCB
Qy	181 MDCSRTKLVIVDIDTYWENGLTIDLBOKLMDADAKLSPHTHARLDSFRQKVEGLTHP	Db	1241 FALTSGDTWTDWOTRSIMACNRGTGKRRKTSALSYSPDIOVSQSERPFTRCB
Qy	301 BONGGCCHLCLSPSPYTCAPCPTVOLQDNGTCKGABRULLARTMRSJLTP	Qy	1361 PAHSSATGPVGIISLFMGGGVYFCVVCYACANGCPEHEVSTCPHPINP
Db	301 BONGGCCHLCLSPSPYTCAPCPTVOLQDNGTCKGABRULLARTMRSJLTP	Db	1361 PAHSSATGPVGIISLFMGGGVYFCVVCYACANGCPEHEVSTCPHPINP
Qy	361 DEDPIVQDPIRHALDVTPLBZVYWDKFRATRAVLDGSCQTJNTEINDPG	Qy	1381 GOSOKRPTJACIGSMSSVSLMGRGCVYDRAFTGASSSSSTKTYLPIUP
Db	361 DEDPIVQDPIRHALDVTPLBZVYWDKFRATRAVLDGSCQTJNTEINDPG	Db	1381 GOSOKRPTJACIGSMSSVSLMGRGCVYDRAFTGASSSSSTKTYLPIUP
Qy	421 IADKLUPIPGTLLGDPITDWQRSIEVKVKASRDVIDQDPMGKAVAKV	Qy	1441 GESOHPPTJACKSMSMSVSLMGRGCVYDRAFTGASSSSSTKTYLPIUP
Db	421 IADKLUPIPGTLLGDPITDWQRSIEVKVKASRDVIDQDPMGKAVAKV	Db	1441 GESOHPPTJACKSMSMSVSLMGRGCVYDRAFTGASSSSSTKTYLPIUP
Qy	481 NPIEBCANLOCOBERTYLWNLGNGMLDLOGKUWGDQKTDKLBVINGTMRIL	Qy	1500 PPSPATPSLNMDDPYSNITATAPRTPYRIGMAPTPCSTVCDSDYASRWKAS
Db	481 NPIEBCANLOCOBERTYLWNLGNGMLDLOGKUWGDQKTDKLBVINGTMRIL	Db	1500 PPSPATPSLNMDDPYSNITATAPRTPYRIGMAPTPCSTVCDSDYASRWKAS
Qy	541 IADKLUPIPGTLLGDPITDWQRSIEVKVKASRDVIDQDPMGKAVAKV	Qy	1501 PPSPATPSLNMDDPYSNITATAPRTPYRIGMAPTPCSTVCDSDYASRWKAS
Db	541 IADKLUPIPGTLLGDPITDWQRSIEVKVKASRDVIDQDPMGKAVAKV	Db	1501 PPSPATPSLNMDDPYSNITATAPRTPYRIGMAPTPCSTVCDSDYASRWKAS
Qy	601 GTNP CADRNGCCSHLCPFPFHATRCGPGLKLSDMKTCIVPEALVFTSRAATHRISL	Qy	1561 KYTLDIANSDDPPPTPSHSQSAECCPPSPATERSYHLLRPPPSCTDSS
Db	601 GTNP CADRNGCCSHLCPFPFHATRCGPGLKLSDMKTCIVPEALVFTSRAATHRISL	Db	1561 KYTLDIANSDDPPPTPSHSQSAECCPPSPATERSYHLLRPPPSCTDSS
Qy	661 ETNNDVAIPVGKEASALDPDVSNHIFTWVLSKTSIRAPMGSSVENVFOLDYP	Qy	1561 KYTLDIANSDDPPPTPSHSQSAECCPPSPATERSYHLLRPPPSCTDSS
Db	661 ETNNDVAIPVGKEASALDPDVSNHIFTWVLSKTSIRAPMGSSVENVFOLDYP	Db	1561 KYTLDIANSDDPPPTPSHSQSAECCPPSPATERSYHLLRPPPSCTDSS
Qy	721 EGMADVMMGKQIYWDGTTRREVARDQFQTVLVRDOPSLAQLDPPTGYWTEM	Qy	1720 RIKYOTAI; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
Db	721 EGMADVMMGKQIYWDGTTRREVARDQFQTVLVRDOPSLAQLDPPTGYWTEM	Db	OC Mammalia; Eutheria; Primates; Catarachini; Hominidae; Homo.
Qy	781 GCKPRAVAFDGTNTLDRVERANDTIDAOULWMTDMIRESNMCGERW	Qy	OC NCBI_TaxID=9606;
Db	781 GCKPRAVAFDGTNTLDRVERANDTIDAOULWMTDMIRESNMCGERW	Db	RN [1]
Qy	841 IADDLPHFGQDVKYTMWHLHSTERAKTSGRNTLQGHDFUDLVEPHSSQ	Qy	RP SEQUENCE FROM N.A.
Db	841 IADDLPHFGQDVKYTMWHLHSTERAKTSGRNTLQGHDFUDLVEPHSSQ	Db	RC TISSUE=Osteoblast;
Qy	901 DGLANDEMHNGQCSQCLLPGSRGCAHSTLDESRNSRSPPTFLLSQSKAISM	Qy	RC MEDLINE=9838278; PubMed=9714764; DOI=10.1016/S0078-1119(98)00311-4;
Db	901 DGLANDEMHNGQCSQCLLPGSRGCAHSTLDESRNSRSPPTFLLSQSKAISM	Db	RA Hey P.J., Twells R.C.J., Phillips M.S., Nakagawa Y., Brown S.D.,
Qy	961 PDDONSPDPLTPLGILRNLTKAIDPLKPKTIVDPSRNSRSPPTFLLSQSKAISM	Qy	RA Kawaguchi Y., Cox R., Xie G., Bugan V., Hammond H., Metzker M.L.,
Db	961 PDDONSPDPLTPLGILRNLTKAIDPLKPKTIVDPSRNSRSPPTFLLSQSKAISM	Db	RA Todd J.A., Hess J.P.,
Qy	1021 NPDROPHDSDIXSLTNTCEATNTINHRLSGRMGVGRDKRPAVINACY	Qy	RT "Cloning of a novel member of the low-density lipoprotein receptor
Db	1021 NPDROPHDSDIXSLTNTCEATNTINHRLSGRMGVGRDKRPAVINACY	Db	RT family,"; Gene 216:103-111(1998).
Qy	1080 1NPDROPHDSDIXSLTNTCEATNTINHRLSGRMGVGRDKRPAVINACY	Qy	RL [2]
Db	1080 1NPDROPHDSDIXSLTNTCEATNTINHRLSGRMGVGRDKRPAVINACY	Db	RP SOURCE FROM N.A.
Qy	1081 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Qy	RP MEDLINE=2129504; PubMed=11400138; DOI=10.1006/geno.2000.6492;
Db	1081 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Db	RA Twells R.C.J., Metzker M.L., Brown S.D., Cox R., Garey C., Hammond H.,
Qy	1141 ISGAMLTLDANVQPLTGLKJWIDQQOMIBERETKTKRPIQPVAHG	Qy	RA Hey P.J., Levy B., Nakagawa Y., Phillips M.S., Todd J.A., Hess J.-P.;
Db	1141 ISGAMLTLDANVQPLTGLKJWIDQQOMIBERETKTKRPIQPVAHG	Db	RT "The sequence and gene characterization of a 400-kb candidate region
Qy	1141 ISGAMLTLDANVQPLTGLKJWIDQQOMIBERETKTKRPIQPVAHG	Qy	RT for 1DDM4 on chromosome 1q13.";
Db	1141 ISGAMLTLDANVQPLTGLKJWIDQQOMIBERETKTKRPIQPVAHG	Db	RT Genomics 72:231-242(2001).
Qy	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Qy	RL [3]
Db	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Db	RP SEQUENCE FROM N.A.
Qy	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Qy	RP PubMed=1150515; DOI=10.1073/pnas.0133721010;
Db	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Db	RA Fujino T., Asaba H., Kang M.J., Ikeda Y., Sone H., Takada S.,
Qy	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Qy	RA Kim D.-H., Ioka R.X., Ono M., Tomoyori H., Okubo M., Murase T.,
Db	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Db	RA Kamataki A., Yamamoto J., Magoori K., Takahashi S., Miyamoto Y.,
Qy	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Qy	RA Oishi H., Nose M., Okazaki M., Usui S., Imaizumi K., Yanagisawa M.,
Db	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Db	RA Sakai J., Yamamoto T.T.,
Qy	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Qy	RT "Low-density lipoprotein receptor-related protein 5 (LRP5) is
Db	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Db	RT essential for normal cholesterol metabolism and glucose-induced
Qy	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Qy	RT insulin secretion.";
Db	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Db	PRO. NATL. ACAD. SCI. U.S.A. 100:229-234 (2003).
Qy	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Qy	[4]
Db	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Db	FUNCTION, PHOSPHORYLATION, AND INTERACTION WITH AKIN.
Qy	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Qy	PubMed=173102; DOI=10.1016/S097-2765(03)00484-2;
Db	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	Db	Tamai K., Zeng X., Liu C., Zhang X., Harada Y., Chang Z., He X.;
RA	1201 IYPTMNDRAKIERAALGSTBERVLFPTGLRIPVALWDTGLKFWADAKRISCD	RA	